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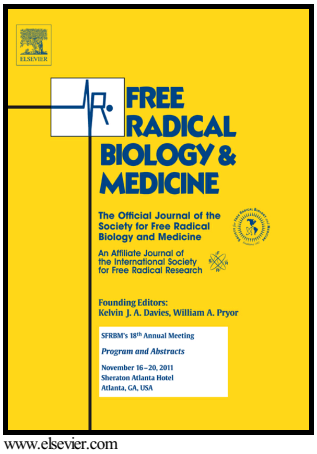

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Abstract

Earlier literature underlines that oxidative stress plays a major role in the pathology of myocardial ischemia-reperfusion (I/R) injury, diabetic cardiomyopathy (DCM), diabetes mellitus (DM), fibrosis and hypertrophy which could adversely affect the normal cardiac function. However, the contributory role of oxidative stress in I/R pathology of heart with pre-existing abnormalities or diseases like DM and DCM remains to be explored. I/R injury was induced in normal (normal diet), DM (normal diet + streptozotocin: multiple low dose of 30 mg/kg) and DCM (high fat diet (40% fat) + streptozotocin: multiple low dose of 30 mg/kg) rat hearts using Langendorff isolated heart perfusion apparatus. Cardiac physiological recovery after I/R was assessed by hemodynamic parameters like LVDP, and LVSP, whereas cardiac injury was measured by tissue infarct size, and apoptosis, LDH, and CK release in coronary effluent. The oxidative stress was evaluated in myocardial homogenate, mitochondrial subpopulation, and microsomes. Reperfusing the ischemic DCM heart significantly deteriorated cardiac physiological recovery and elevated the cardiac injury (infarct size: 60%), compared to the control. But in DM heart, physiological recovery was prominent in the initial phase of reperfusion but deteriorated towards the end of reperfusion, supported by less infarct size. In